A STUDY OF LOWER GI MALIGNANCIES

Dissertation submitted in partial fulfillment of regulation for the award of M.S. Degree in General Surgery

(Branch I)

The Tamilnadu
Dr. M.G.R. Medical University
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CERTIFICATE

Certified that this is the bonafide dissertation done by **Dr. P. JOHN PAUL** and submitted in partial fulfillment of the requirements for the Degree of M.S., General Surgery, Branch I of The Tamilnadu Dr. M.G.R. Medical University, Chennai

Date : Unit Chief

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DECLARATION

I solemnly declare that the dissertation titled **"A Study of Lower GI Malignancies"** was done by me from 2006 onwards under the guidance and supervision of **Prof. Dr. P. GOVINDARAJ, MS., MCh.,**

This dissertation is submitted to the Tamilnadu Dr. MGR Medical University towards the partial fulfillment of the requirement for the award of MS Degree in General Surgery (Branch I).

Place : **Dr. P. JOHN PAUL**

Date :

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CONTENTS

INTRODUCTION

Lower GI malignancies though more common in the west are on the increase in our country for the past decade [44]. Few topics in cancer research have engendered more excitement than the recent discovery of identifiable genetic defect in patients with inherited as well as sporadic form of colorectal carcinoma & the ideal goal of treatment in lower GI malignancies is the eradication of disease with preservation of anatomic & physiological function.

There are evidences that neoplastic disease has affected humans since prehistoric times. Mummies from pre – Columbian, Peru of 2400 years ago as well as Egyptian mummies from 3000 B.C. have metastatic skeletal deposits. It was Hippocratic (460 – 370 B.C.) who first proposed a theoretical framework to explain cancer invasion.

The cellular etiology of cancer was first described by Johannes Peter Muller in 1828. The following year Joseph Calrude Recaemer proposed that invasion and distant spread were the result of translocation of cells and he coined the term metastasis. The first successful resection of colonic growth was performed by Reynoard Lyons in 1823.

After Billroth, Czerny and Mickulicz, the pioneers in abdominal surgery familiarized, the technique of intestinal resection and anastamosis & increasing number of colonic resections were attempted. The combined

operation involving abdominal and perineal phases for excision of the rectum was first performed by Czerny (1883). But it was undoubtedly the work of Emest Miles (1908) that established the abdomino perineal operation.

Cuthberk Dukes (1935) classified carcinoma of rectum into 3 stages and explaining macroscopic variations [1]. These are widely used by pathologists with minimal changes even now for colorectal cancer staging .Paul of Liverpool (1895) and Mikulicz of Brestan (1903) devised extra peritoneal resection of carcinoma colon and popularized the technique in America.

Halstead (1895), Shoemaker (1921) Ranken (1928) and Wangenstein (1940) described various methods of anastamosis by which it was hoped to carry out resection and anastamosis in an entirely sterile manner without opening the bowel lumen till union was completed. But it was later pointed out by Moyhnian that the factor responsible for sepsis is not contamination during the operation itself but subsequent leakage.

Whipple (1931) and Turner (1937) favoured intraperitoneal resection with temporary caecostomy in order to relive the tension on the suture lines. Devine (1931) developed preliminary defunctioning colostomy which helped mechanical cleansing of the distal bowel.

After advent of strong intestinal antiseptics, reliance was placed on them entirely and a primary colostomy was entirely omitted. Lloyd Davis

Morgan and Yollinger (1953) carried out resection with immediate anastamosis without any form of proximal decompression. In their series of 109 cases, there were only 3 postoperative deaths and none of them was due to sepsis.

In recent years, the trend is towards preparation with mechanical cleansing using balanced salt solutions containing osmotic purgatives in them with antibiotics, orally or IV [24]. This requires only single day preoperative preparation [16].

Surgical resection remains the mainstay of treatment for Lower GI malignancies [2]. Radiotherapy and chemotherapy are used as adjuvant therapeutic options. Turnball at the Cleveland clinic recommended a no touch technique in which vascular and mesenteric division was first undertaken, thereby isolating the tumour.

The role of gene and their abnormality are being studied extensively and has given us the *adenoma to carcinoma* model due to accumulation of various genetic defects in the form of deletion, translocation etc. These may help us to find appropriate diagnostic tool to look for such aberration early, preventing cancer progression..

Colorectal surgery has advanced a lot with introduction of endo GIA staplers. Especially for sphincter saving procedures for carcinoma rectum[31] with the advent of the laparoscope , laparoscopy assisted resection procedures are also being carried out [13].

AIM

The aim of the study is,

1. To study the clinical presentation of lower GI malignancies, including the incidence of emergencies.

2. To study the macroscopic and histopathological types of lower GI malignancies in our institution.

3. To evaluate various types of surgeries for lower GI malignancies being performed in our institution.

REVIEW OF LITERATURE

SURGICAL ANATOMY OF LOWER GI TRACT

The small intestine is 6 meters in length. This includes the lower mobile part beyond the DJ flexure (proximal 2/5 is jejunum & distal 3/5 is ileum) .The large intestine extends from end of ileum to anus and comprises of the caecum (with appendix) colon, rectum, anal canal, measuring between 110 – 170 cm in length on an average 135 cm long.

EMBRYOLOGY:

The small intestine develops from midgut. The large intestine develops from both mid gut and hind gut. Midgut portion extends from caecum to the proximal 2/3 of transverse colon supplied by superior mesenteric artery. Hindgut, from distal 1/3rd of transverse colon to proximal anus is supplied by inferior mesenteric artery. The distal anal canal is ectodermal in origin & is supplied by internal pudendal vessels.

Large guts starts developing by fifth week of gestation and is completed by eight week of gestation when the anal membrane ruptures. During sixth week, migration and midgut rotation occurs over 4 weeks assuming final anatomic position by 10 week of gestation.

ANATOMY

THE JEJUNUM & ILEUM :

The Jejunum & Ileum measure nearly 5 to 5.5 meters, of this proximal 2/5 is jejunum & distal 3/5 is ileum . The jejunum & ileum are supplied by the branches of superior mesenteric artery.. Venous drainage is through superior mesenteric vein. The lymph vessels accompany the veins & end in 3 sets viz.,1.distal set , 2. intermediate set.,3. proximal or superior mesenteric nodes.

THE CAECUM :

The caecum, lies in the right iliac fossa, approx.7 cms in length and width. Proximally becomes ascending colon at it's junction with terminal ileum guided by a valve which prevents reflux .

It lies on iliac and psoas muscle and on genitofemoral, lateral cutaneous nerve of thigh. It's exact position is variable, may extend into true pelvis. It is almost completely enveloped by peritoneum often attached to iliac fossa medially and laterally.(Fig.1)

THE ASCENDING COLON :

It varies from 10 - 20 cms. It lies on iliac – muscle, iliac crest, quadrates lumborum and crossing lateral cutaneous nerve of thigh, ilioinguinal and iliohypogastric nerves, usually covered with peritoneum on all 3 sides except posteriorly. Where it is fixed to post abd wall,

sometimes it may be fixed by a short mesentery. It ends at hepatic flexure where it turns left on the lower portion of right kidney.

THE HEPATIC FLEXURE :

At this point the colon turns sharply medially and slightly forwards downwards just below right lobe of liver and overlapped by it, posteriorly lies on lower aspect of right kidney.

THE TRANVERSE COLON :

It the longest of all, varying from 40cm to 70cm in length extending from hepatic flexure to splenic flexure forming a dependent loop between the points. It is suspended by transverse mesocolon which is attached to descending part of duodenum, lower aspect of body of pancreas and anterior surface of left kidney. It contains middle colic vessels and branches of left colic artery, right colic artery and lymphatics.

THE SPLENIC FLEXURE :

It is junction of transverse colon and descending colon in left hypochondrium. The turn is so acute, transverse colon overlaps it anteriorly. Related to spleen and pancreatic tail superiorly and front of left kidney, medially attached to diaphragm level with X, XI, ribs via phrenicocolic ligament which lies below the antero lateral pole of spleen.

THE DESCENDING COLON :

It extends from splenic flexure to rim of true pelvis close to inguinal ligament from where it continues as sigmoid measuring 25 cm. Attached to posterior abdominal wall by peritoneum, in left paravertebral gutter to left iliac fossa. It rests on the same muscle and related to the same nerve as ascending colon.

At antero superior iliac spine it turns medially, superior to inguinal ligament and lies on femoral nerve, psoas muscle, genital vessels, becomes sigmoid colon anterior to external iliac vessels.

THE SIGMOID COLON :

It is the most variable part measuring 40 – 80 cm in length. Extends upto rim of true pelvis where it becomes the rectum, suspended by sigmoid mesocolon, a long mesentery with short base.(Fig.4)

The position, shape, vary very much depending on its length, length and mobility of its mesocolon, the degree of distension,condition of rectum, bladder, uterus.

THE RECTUM :

It lies in the true pelvis measuring about 12 – 15 cm with a diameter of 4 cm when empty. It is dilated in the lower part to form rectal ampulla.

It follows curve of sacrum and coccyx, runs anteriorly, inferiorly to central perineal tendon lies on levator ani muscles, anococcygeal

ligament. It ends posterior to central perineal tendon and to the apex of prostate in male by turning posteriorly and inferiorly as anal canal.

It follows the curve of sacrum & coccyx in sagittal plane. In coronal plane it is shaped giving rise to prominent folds in the lumen known as Houston's valves.

The relationship of pelvic peritoneum to rectum is of considerable surgical importance. The upper third has a complete peritoneal investment except for a thin strip posteriorly where peritoneum is reflected as the two leaves of thick mesorectum. As rectum descends into pelvis, the uncovered portion becomes wider until only anterior aspect has a peritoneal coat in middle 1/3 of rectum. This peritoneum gets reflected forward in the bottom of rectovesical pouch or rectouterine pouch leaving lower third of rectum extra peritoneal.

Posteriorly the parietal pelvic fascia is thickened to form fascia of Waldeyer's separating rectum from sacrum coccyx, blood vessels, and nerves. Anteriorly separated by the fascial layer known as Denon villier's fascia.

The upper 2/3 of rectum is separated from pelvic fascia by posterior cushion of areolar tissue which becomes circumferential below rectovesical / rectouterine pouch carrying blood vessels and its lymphatics known as mesorectum.

THE ANAL CANAL :

It is about 4 cms long with anterior wall shorter than posterior one. When empty its lumen is a triradite longitudinal slit, posteriorly anococcygeal ligament separates it from coccyx while inferiorly perineal body separates it from membranous urethra, penile bulb or lower vagina laterally, ischiorectal fossa. It's whole length is surrounded by sphincters which keeps it closed.

The mucosa of anal canal consists of an upper mucosal and lower cutaneous part, the junction being marked by line of anal valves about 2 cm from anal orifice known as Dentate line .

BLOOD SUPPLY AND LYMPHATIC DRAINAGE :

These two are important subjects in relation to malignancy and its treatment.

BLOOD SUPPLY :

The main arteries supplying the jejunum, ileum, colon, rectum are superior mesenteric artery, inferior mesenteric artery, middle, inferior rectal arteries. The jejunum, ileum, caecum, ascending colon, hepatic flexure and proximal two thirds of transverse colon derive blood supply from superior mesenteric artery originating from aorta at L1 level via ileo colic , right colic, middle colic vessels. The distal third of transverse colon, splenic flexure, descending colon, sigmoid and upper third of

rectum via inferior mesenteric artery rising from aorta at L3 level via left colic, sigmoid branches and superior rectal artery. The distal two thirds of rectum, anal canal get blood supply via middle, inferior rectal artery of internal iliac artery, internal pudendal artery.

The jejunal & ileal branches form multiple arterial arcades to supply the jejunum & ileum. The main colic arteries proceed to colon and bifurcate to from branches which unite to form arcades an inch or so from mesenteric border, so that a continuous chain of communicating vessels is formed. This is the marginal artery of Rolando , from it, the ultimate branches supply the colon. (Fig.2)

These branches ramify between and supply muscular layers, divide into small submucosal rami and enter the mucosa. The marginal artery is responsible for bringing the area of supply of the superior mesenteric artery into communication with that of inferior mesenteric by connecting the descending branch of the middle colic with the ascending branch of the left colic by means of long anastomosis of colon.(Fig. 3)

The venous drainage follows its arterial blood supply empties into portal venous system. The inferior mesenteric vein diverges from artery and passes behind pancreas to drain into splenic vein. Splenic vein and superior mesenteric vein join behind neck of pancreas to form portal vein which drains the blood into liver.

LYMPHATIC DRAINAGE

The entire jejunum & ileum drain to the 3 sets of nodes situated in the distal ., intermediate & proximal positions., The colon and rectum are drained by a large number of lymph node numbering 70 – 100 which are present as a series draining into a principal nodal group.

A. INTRAMURAL LYMPHATICS :

Throughout the jejunum, ileum., colon & rectum, there is a rich continuous lymphatic plexus in the submucous or subserous layers of the bowel wall and are inter connected and drain into extramural lymphatics.

B.EXTRAMURAL LYMPHATICS OF COLON :

These consist of lymphatic channels and glands which are divided into 4 groups.

1.Epicolic ,2. **Paracolic** , 3.**Intermediate Colic, 4.Preterminal colic**

C.EXTRAMURAL LYMPHATICS OF RECTUM:

Likewise lymphatics drain to pararectal group in contact with wall of rectum, hence to intermediate group around main arterial for (superior rectal artery) and hence to nodes near origin of main vessel.

From upper half of rectum; lymphatic flow to pararectal nodes from thence to superior rectal nodes which drain to nodes in lower sigmoid colon thence to nodes along inferior mesenteric artery .

Laterally along middle hemorrhoidal vessels on either side to ischiorectal fossa and thence to internal iliac glands via inferior rectal and internal pudendal vessels (above mucocotaneous junction) .

Lymphatics of anal canal, below Dentate line descend to anal margin, curves laterally to reach medial superficial inguinal nodes.

SURGICAL PHYSIOLOGY OF LOWER GI TRACT

The complex process of digestion & eventual absorption of nutrients - carbohydrates, proteins & fats, water, electrolytes., & vitamins., is the main role of small intestine., Nearly all food is absorbed by the small intestine except for indigestible cellulose.

The large intestine receives the ileal contents, absorbs water and electrolytes and acts as a reservoir for the fecal matter until it is suitable to be discharged through the anus. It was calculated by Smidday et al (1960) that about 800 – 1000 ml of fluid enters the large intestine each day and 150 ml of this is passed in the feces. Complete loss of colonic and rectal function occurs during ileostomy and total proctocolectomy procedures. The importance of terminal 30 cm of ileum was emphasized by Lillcher and Wangensteen (1956) urging conservation if possible.

When ileocaecal valve is removed in right hemicolectomy bowel function is altered to give an increased stool frequency upto 4 times a day. This is due to colonic reflux with bacterial colonization of small bowel and loss of regulating valve. After left hemicolectomy only a slight increase in stool frequency occurs.

INCIDENCE / EPIDEMOLOGY

It is a dynamically changing disease entity due to multifactorial reasons. It is predominantly a tumour of old age > 50 yrs and can occur in young individuals (genetic inheritance)

90% of carcinoma occurs in people more than 50 yrs old [4]. There is a definitive male preponderance (more in rectal carcinoma than colonic and small bowel tumours) averaging 1.3 – 1.8 : 1 sex ratio [3].

The incidence of cancer is much higher in western countries suggesting environmental and genetic factors. The frequency has been increasing in our country over the last few years possibly related to changing dietary, social habits [44].

It is observed from earlier statistics Smiddy, Goligher (1967) that recto sigmoid accounts for more than half cases of colorectal cancer. Now there is a progressive trend towards disease of right colon and fewer left colon and rectal cancers. (Cardy B Person, 1977, Steele GD 1979). In this study, there was more cases involving the left side [31].

Carcinoma of Rectum accounts of nearly one third of all cancers, followed by carcinoma of sigmoid colon, cancer caecum and recto sigmoid junction – followed by others. In order of decreasing frequency in others are transverse colon, ascending colon, descending colon, splenic flexure and hepatic flexure (Bailey & Love)

ETIOPATHOGENESIS:

Numerous risk factors and associated conditions viz., FAP, HNPCC, Peutz-Jeghers Syndrome, Crohn's disease, Ulcerative Colitis, celiac sprue, smoking, alcoholism, consumption of red meat have been found to be associated with increased risk for small bowel tumours. The exact cause of colorectal cancer is not known precisely, with recent work providing that a complex interaction between genetic makeup and the environment in which the subject resides determines the incidence [42]. Majority of neoplasms are adeno carcinomas, with malignant melanoma, squamous cell carcinoma being rare variants in anal canal.

ETIOLOGY:

Genetic Predisposition:

Approximately 10 – 15% of colorectal cancer is familial. 1 in 200 person may carry those genes, with mutation in these genes responsible for 15% of sporadic cancers [43].

Hereditary non polyposis colon cancer:

Warthin in 1913 recognised increased incidence of cancer in families with lynch characterizing these cancer – prone families which was not associated with multiple polyps, hence the name.

Two distinct clinical presentations were made out,

Lynch Syndrome I - Site specific proximal colon cancer in the family.

Lynch Syndrome II - Characterised by the development of colorectal, endometrial, gastric, upper urinary tract, ovarian and other malignancies.

Familial adenomatous polyposis: It is an autosomal dominant disorder, diagnosed when a patient has more than 100 adenomatous polyps in colon or when a member of an FAP family has any number of colonic adenomas detected. The basic defect is due to mutation in APC gene located in chromosome 5 q21 locus [33]. All patients with this defect will develop colonic cancer if left untreated, hence recommendation state periodic colonoscopic examination and prophylactic polypectomy with HPE to rule out carcinoma or prophylactic proctocolectomy. One marker is congenital hypertrophy of retinal pigmented epithelium seen in 70 – 80% by opthalmoscopy.

Variants of FAP are,

Attenuated adenomatous polyposis coli. The patients have less number of polyps with same high risk of malignancy.

Hereditary flat adenoma syndrome develops small adenoma, less than 100, frequently dysplatic prone for malignant change.

Others in spectrum of hereditary polyposis syndrome are

A. Gardner's Syndrome – Colonic polyposis, epidermal inclusion cyst, osteomas of bone, upper GI Tumours.

B. Turcot's Syndrome – Colonic polyps with Brain Tumours (medulloblastoma)

Environmental Factors:

Diet:

Proposed as a significant etiologic factor, fats suggested to be toxic to intestinal mucosa with saturated fats to be more carcionogenic. Studies have shown that people whose diet contain <5% fat have lower incidence of colorectal cancer.

Other compounds suggested to be carcinogenic are fecapentanes produced by gutflora, 3 ketosteroids metabolic product of cholesterol, pyrrolysis product formed by smoking or deep frying meat products, rice, etc. Even increased bile acids are thought to be carcinogenic.

Deficiency of compounds such as calcium, vitamin D, A, Vitamin C, tocopherol, selenium and dietary fibre may be associated with increased incidence due to decreased protective effects by them.

This dietary role is proved by people who immigrate from low risk regions to high risk region acquiring high risk in a generation time.

Carcinogens:

No clear relationship has been established between specific carcinogens and small bowel, colorectal cancer. Potential agents under study include Bile acids, food additives, smoking, alcohol, ionizing radiation, oxygen free radicals may serve as promoter or stimulants to altered gene development.

PRE MALIGNANT CONDITIONS:

Ulcerative Colitis:

Overall incidence of cancer in patient with pan colitis is 1% per year after 10 yrs by 20 yrs, Dysplasia is a precursor to cancer. But implication of polyps, adenomas is not fully clear. Once dysplasia is identified removing colon is the most effective method to prevent cancer. Risk of cancer in dysplasia is upto 30%

Crohn's Disease:

Overall incidence of cancer is 7% over 20 yrs. Risk of cancer is high in bypassed segments, in fibrotic narrowing, and sites of stricturoplasty.

Previous malignant disease:

Patient who underwent treatment for cancer large bowel have three fold increased risk of second endorectal malignancy.

Polyps:

Adenomatous polyps more than 2 cm in size are prone for malignancy. Patient with multiple polyps, villous adenomas when compared to tubular ones have increased incidence [15].

Influence of hormones:

The lining of colon is exposed to a variety of endogenous substances exerting tropic effects on mucosa. Gastrin appears to be most directly related to colonic carcinogenesis.Leptin acts as a mitogenic and antiapoptotic factor for Colonic cancer cells [28]

Elevated levels have been demonstrated in patients with colorectal cancer. Other growth factors associated are transforming growth factor (TGF), Bombesin, IGF.

Others:

Pelvic Radiation : Supportive, but inconclusive evidence found between radiation and intestinal tumours. Over all risk is very small.

Previous Non cancer Surgery:

Cholecystectomy, uretero sigmoidostomy patients have increased incidence.

PATHOLOGY AND SPREAD

In initial stages, cancer of lower GI tract takes the form of localised area of thickening of the normal mucosa or a hard nodule in a preexisting adenoma or villous papilloma.

There are 4 distinct macroscopic types. (Fig. 5)

A. Polypoid or cauliflower growth (Fungating / Exophytic):

This produces a large fungating mass which projects into the lumen of the bowel and is not usually associated with much infiltration of the intestinal wall more commonly seen in proximal colon, ascending colon, caecum, etc.

B. Annular or constricting or circumferential growth:

These lesions extend around bowel wall and the bowel looks as if deeply constricted by a string around it., more commonly seen in left sided growths i.e., descending colon, sigmoid colon.

C. Ulcerating Growth:

Presents as a typical malignant ulcer and this infiltrates the bowel wall producing deformity and narrowing of the lumen.

D. Stenosing or Diffusely infiltrating growth:

This corresponds to linitus plastica of stomach and produces thickening of the intestinal wall usually extending for at least 2 – 3 inches

and for most part covered with intact mucosa., more common in the left side growths.

While all consider the above types as the 4 macroscopic appearances, Duke classified adenocarcinomas which produce abundant mucin as.,

E. Colloid Carcinoma : This forms a bulky growth with getatinous appearance and may or may not be associated with ulceration and infiltration.

Histologic Types:

The most common type is adenocarcinoma (Fig. 6 A, 6 B), the other types are:

1. Mucinous adenocarcinoma 2. Signet ring cell adenocarcinoma
3. Squamous cell Carcinoma 4.Adenosquamous Carcinoma
5. Undifferentiated Carcinoma

Other types are Carcinoid tumours

Non epithelial tumours

Degree of Differentiation:

In general papilliferous growths tend to be better differentiated than the ulcerating or infiltrating type.

Broders grading:

Broders designated adenocarcinoma into 4 grades based on percentage of differentiated tumour cells.

Grade I : Well differentiated tumours, closely resembling an adenoma

Grade II : Tumour cells more crowded together but still arranged in fairly regular pattern.

Grade III : Less differentiated and arranged in irregularly folded rings.

Grade IV : Anaplastic cells which did not form glandular structures at all.

Mucoid tumours vary considerably and were graded separately by Duke.

Duke's Grading:

Duke considered arrangement of cell and evolved into new three grade system.

Grade I : Well differentiated , well formed tubules with least nuclear pleomorphism and mitosis.

Grade II : Moderately differentiated.

Grade III : Least differentiated – occasional glandular structure., more pleomorphic cells, large number of mitosis.

SPREAD OF LOWER GI MALIGNANCIES

Most of our knowledge of spread was due to studies by Dukes (1930) Gordon – Watson.

A. LOCAL INVASION:

First spread after initial mucosal growth was to protrude into the lumen. Lateral invasion was more in transverse direction leading to circumferential growth. (Duke, Black and Waugh)., Mural penetration leads to local failure and peritoneal seeding. Additional spread is via perineural spaces with invasion reaching as far as 10 cm from the primary tumour [32].

B. LYMPHATIC EXTENSION:

Lymph nodal metastasis occurred only after tumours have penetrated into first set of nodes. Lymphatics spread occur in an orderly fashion via the three echelons of nodal systems.

Retrograde spread occurs on blockage of central nodes. The risk of lymphatic spread increases with increasing tumour grade. As spread follows the course of blood vessels supplying the carcinomatous region, appropriate fields of excision is worked out by reference to arterial supply.

C. HEMATOGENOUS SPREAD:

The liver is the primary site of hematogenous spread., followed by lungs. As venous drainage of rectum is via dual systems, liver and lung are involved primarily depending on site of tumor origin in rectum.

Batsons vertebral plexuses represent another way of blood spread of metastasis to bone and CNS.

D. IMPLANTATION:

It refers to release of tumour cells from primary site and their deposition on another surface, it can occur transluminally, transperitoneally after seroal invasion and during surgical manipulation diminishing curative resection and increasing local failure rate, and distant deposits.

STAGING AND CLASSIFICATION

Duke presented the first original staging for rectal carcinoma which is widely used now to stage both colorectal tumours.

DUKE STAGING (1930):

A - Growth limited to colonic / rectal wall but not through

B - Growth penetrating through Bowel Wall

C - Involvement of lymph node regardless of extent of Bowel wall penetration

1935 Modification:

A, B as same.

- C1 - Locally positive nodes

C

- C2 - Positive nodes at point of ligature

ASTLER AND COLLINS MODIFICATION:

A - Limited to Mucosa

B1 - Extending into, but not through propria with uninvolved nodes.

B2 - Extending into and through mucularis propria, with uninvolved nodes

C1 - Extending into, but not through muscularis propria with involved nodes

C2 - Extending through M.Propria with involved nodes.

This staging allowed separation of wall penetration and nodal status.

TNM CLASSIFICATION FOR COLONIC CANCERS:

T **-** ***Tumour Stage***

T1 - Into submucosa

T2 - Into muscularis propria

T3 - Into pericolic fat but not breaching serosa

T4 - Breaches serosa or directly involving another organ

N **-** ***Nodal stage***

N0 - No Nodes involved

N1 - 1 or 2 nodes involved

N2 - 3 or more nodes involved

M **-** ***Metastasis***

M0 - No metastasis

M1 - Metastasis

Ly **-** ***Lymphatic invasion***

L0 - No lymphatics involved

L1 - Lymphatics involved

V **-** ***Venous invasion***

V0 - No vessel invaded

V1 - Vessels invaded

R **-** ***Residual tumour***

R0 - No residual tumour

R1 - Margins involved, residual tumour present

TNM staging for Rectal cancer

T1 - Tumour invasion through muscularis mucosa but not into muscularis propria

T2 - Tumour invasion into but not through muscularis Propria

T3 - Tumour invasion through muscularis propria but not through serosa

T4 - Tumour invasion through serosa or mesorectal fascia

N0 - No nodes involved

N1 - 1 – 3 nodes involved

N2 - 4 or more nodes involved

M0 - No metastasis

M1 - Distant metastasis

TNM staging for anal malignancies

Primary Tumour (T)

Tx - Primary tumour cannot be assessed

T0 - No evidence of primary tumour

Tis - Carcinoma in situ

T1 - Tumour < 2.0 cm in greatest dimension

T2 - Tumour >2.0 cm but not > 5.0 cm

T3 - Tumour > 5.0 cm

T4 - Tumour of any size that invades adjacent organ(s)

Regional Lymph Nodes

Nx - Regional lymph nodes cannot be assessed

N0 - No regional lymph node metastasis

N1 - Metastasis in perirectal lymph nodes

N2 - Metastasis in unilateral internal iliac and / or inguinal lymph nodes

N3 - Metastasis in perirectal and inguinal lymph nodes and or bilateral internal iliac and / or inguinal lymph nodes

Distant Metastasis (M)

Mx - Distant metastasis cannot be assessed

M0 - No distant metastasis

M1 - Distant metastasis.

CLINICAL FEATURES

There are 3 main ways in which carcinoma of lower GI malignancies may present.

A. As non emergency cases with insidiously developing chronic symptoms chiefly affecting bowel function and general health.
B. As emergencies with perforation / obstruction of bowel with or without peritonitis.
C. Non specific symptoms

The common symptoms comprise the following [44]:

- ❖ Altered bowel habits in the form of alternating constipation and diarrhea, tenesmus etc.,
- ❖ Pain
- ❖ Sense of incomplete evacuation
- ❖ Bleeding per rectum
- ❖ Mucus per rectum
- ❖ Mass per abdomen
- ❖ History of haemorrhoids
- ❖ Unexplained anaemia
- ❖ Abdominal pain / dyspepsia
- ❖ Loss of weight, asthenia, impairment of general health
- ❖ Loss of appetite
- ❖ Flatulent distension

- ❖ Acute on chronic bowel obstruction
- ❖ Bowel perforation / peritonitis

Fairly definite correlation between site, type of growth and symptomatology occurs. Carcinoma of left colon and rectosigmoid present early due to stenosis / circular growth whereas right colon growth present late. Local spread may present with related symptoms like rectovaginal fistula, recto vesical fistula, urinary obstruction, hemorrhoids etc.,

Carcinoma of the small bowel:

This includes adenocarcinomas, carcinoid tumours, malignant GISTs, and lymphomas which present with mainly pain, weight loss, anemia, diarrhea, tenesmus, passage of large amount of mucus, lower GI bleed, malena and acutely as obstruction or perforation.

Carcinoma of caecum and ascending colon:

Most present with anemia, severe and unyielding to treatment. A palpable tumour may be present. Sometimes discovered unexpectedly at operation for acute appendicitis for an appendicular abscess failing to resolve. Sometimes carcinoma of caecum can be the apex of an intussusception presenting with intermittent obstruction [49].

Carcinoma of transverse colon:

It may be mistaken for carcinoma of stomach due to position of tumour together with anemia and lassitude.

Carcinoma of left side of colon:

Majority of tumours occur in this location. They are usually of stenosing, annular, ulcerative type. The main symptoms are those of increasing intestinal obstruction. Colicky or constant aching pain may be the only symptom. Alteration in bowel habits, palpable lump, abdominal distension are the other symptoms.

Carcinoma of rectosigmoid:

Colicky pain, tenesmus, are the usual symptoms., Rectal carcinoma presents usually with bleeding per rectum, sense of incomplete defecation tenesmus and alteration in bowel habits. Subacute intestinal obstruction may induce colicky pain abdomen. Pain in the back, sciatica indicate local sacral plexus involvement. History of piles may be the presenting complaint. Symptoms pertaining to local or distant spread may be the initial presentation in some. May present as fistula in ano single or multiple, discharging pus.

Carcinoma of Anal Canal:

Commonly presenting early with bleeding P/R, pain perianal region, mass perianal region. Patient may present with constipation, may

also present as fistula in ano., may be mistaken and left behind giving rise to obstruction, intestinal distension.

Lower GI Malignancies presenting with acute obstruction:

In about 1/5 of patients, complete obstruction occurs either an aggravation of chronic affairs or as an acute on chronic obstruction or may present one day suddenly as pain, abdominal distension, the narrowed lumen plugged by a fecolith, hard stools, undigestable fibres or tumours.

Chronic obstruction is more commonly encountered with left colonic carcinoma rather than right sided lesions. Constricting stenosing types seen more on left side and the faecal nature produce greater occurrence of acute obstruction.

Symptoms:

Complete obstruction of lower GI tract is often entirely insidious in onset. If acute on chronic variety with development of acute obstruction, the patient finds he is constipated without passing motion for 2 – 3 days despite use of laxatives. He is not able to get rid of flatus and with progressively increasing abdominal distention. This state may be prolonged with slow increase in abdominal discomfort and distension over a period of 6 – 7 days before finally presenting to the hospital.

Carcinoma colon with Bowel Perforation:

Colonic perforation occurs in 3 – 8% of patients with colo rectal cancer. It is often the result of obstructive symptoms prolonged, followed by perforation. The common belief was that perforation takes place in a stercoral ulcer usually in the caecum and less commonly closer to growth. But in a series of 115 cases of carcinoma of large intestine presenting with perforation only 20 cases of this type were found, while majority was proximal to an obstructing cancer producing localized abscess or generalized peritonitis.

Obstructed cases with perforation of stercoral ulcer:

These patients are usually desperately ill with abdominal distension, diffuse tenderness, vomiting, gross dehydration and electrolyte imbalance. The underlying carcinoma may / may not be palpated or suspected from clinical observation. In majority of cases, unless supportive measures are done to rapidly stabilize the patient, patient condition rapidly deteriorates to end fatally [50].

Unobstructed cases with perforation:

Most of these patients are also gravely ill and shows signs of general peritonitis, but owing to the absence of previous obstruction, contamination is less. In some, perforation results in localized peritonitis, abscess formation, producing diagnostic confusion.

If a sigmoid colon growth perforates, this easily mimics a diverticulitis with localized peritonitis with abscess formation. Case recordings are present of a left colonic carcinoma perforation and subsequent development of subcutaneous abscess of abdominal wall (Smith 1963) and subcutaneous emphysema with caecal carcinoma, the presentation may mimic an appendicitis or abscess formation.

INVESTIGATIONS

Routine laboratory investigations to be done including complete hemogram, B. Urea, B. Sugar, Sr.Creatinine, Urine routine examination, E.C.G., Chest X ray, X - ray Abdomen, including Liver Function Tests to ascertain general condition of the patient and treatment of these abnormalities to allow treatment of cancer.

Definitive test for detection / diagnosis of lower GI malignancy

Test for occult blood:

Guaiac test – detects 20 mg hemoglobin per gram or 20 ml blood / day. High false positive results occur due to presence of other oxidizing agents.

Immuno fluorescent test – detects conversion of hemoglobin to fluorescent porhyrins, detects 5 – 10 mg hemoglobin / gm of stool. False positives - prohibitively high.

Rectal examination : reaches approximately 8 cms. 20% colorectal cancers palpated and their degree of fixation evaluated.(Fig. 7)

Proctosigmoidoscopy:

Rigid one is 2 cm in diameter 25 cm long, reaching 20 – 25 cm from dentate line, detect 20 – 25% of tumours, used for screening low risk adults less than 40 yrs with enema to prepare the patient before examination. (Fig. 8)

Flexible sigmoidoscopy:

It measures 60 cm in length reaching upto splenic flexure and identify 50% of tumours can be done after 2 – 3 prep enemas, used only as a diagnostic tool with biopsy taking, with no therapeutic manouvers recommended for people over 50 yrs.

Colonoscopy:

Allows visualization of mucosa of entire colon, rectum and usually terminal ileum.(Fig.12). Measuring about 160 cm in length, it allows biopsy, polypectomy, heamorrhage control & stricture dilatation. Major complication rate < 0.2% with (Bleeding, anesthetic complications, perforations).[21] (Fig. 9)

Recommended annually or once in 2 yrs in patients with high risk of colorectal malignancy with detection of upto 100% of all tumours by a experienced one.(Fig.33,34,35)

Contrast Studies:

Apart from plain Barium meal series and barium enema, an air contrast barium enema is 90% sensitive in detecting polyps >1cm. Risk of perforation < 0.02% with barium enema. Water soluble contrast can be substituted for barium in patients suspected of perforation.(Fig.10,11)

Double contrast barium enema in conjunction with flexible sigmoidoscopy will be a cost effective alternative in patients who do not tolerate colonoscopy.

Imaging techniques:

These are important in the evaluation, staging and follow up of colorectal cancer patients.

USG Abdomen:

Detects early mass lesions involving the lower GI tract & hepatic metastasis [30] .

CT Scan:

It allows preoperative evaluation of abdominal cavity to identify metastasis, integrity of urinary tract and staging the primary tumour lesion especially rectal cancer [48]. Angio CT is 95% sensitive to detect metastases especially liver. Also 60 – 80% rectal wall invasion and 75% of lymphadenopathy can be made out by CT Scan [32]. CT colonography is replacing barium enema for incomplete colonoscopy. Double Contrast CT with CECT with increased cutting slices have improved the efficacy of diagnostic methods.(Fig 13,14).

MRI Scan:

Phased array pelvic coils are used to detect / stage rectal cancer. MRI is more specific for differentiation of cancer from normal tissue and fibrotic scar. It is also more specific to make out liver metastases especially resectable ones.

Positron – Emission Tomography:

It is still investigational, and may be most helpful in evaluating recurrent tumour in pelvis when dense scar tissue is present, other major advantage is to identify extra hepatic / Intraperitoneal disease before surgery.

Trans rectal Ultrasound:

Also known as endosonography of rectum used to stage rectal cancer., USG image allows clear delineation of rectal wall layers, depth of invasion, adjacent origan involvement.

Positive predictive value T < 1 cm - < 50%

> 1 cm – 70%

Endoluminal USG:

Colonoscopic USG in available, allowing determination of depth of invasion, attachment to adjacent structures, mesenteric lymph nodal spread. Also used for USG guided LN Biopsy.

Tumour Markers:

CEA Assay:

In 1965 Gold and Freedman, isolated a specific antigen in adenocarcinoma of the endodermally derived epithelium of the GIT. They were also able to demonstrate the same antigen in embryonic and fetal digestive tract tissue up to the end of the first 6 months of pregnancy

Circulating antibodies to it were found in the sera of a high proportion of women throughout pregnancy normal range, varies from 0 – 2.5 ng/ml [22] .

Thomas et al (1960) developed RIA capable of detecting minute quantities of CEA in serum. Moore et al (1971) further investigated CEA and noted that apart from colorectal carcinoma it was found in carcinoma pancreas and other GIT cancers, bronchogenic carcinoma, alcoholic liver disease and uremia.

The main use of CEA assay will be the monitor patients who had radical surgery for detecting recurrence of disease, persistence of the lesion in metastatic form; other tumour makers include CA 19-9 and CA-50.

AFP:

Alfa feto protein levels indicate basically liver pathology. Levels have been found to be increased in metastatic diseases.

Immunoscintigraphy

A recent development in diagnostic tools utilizes the radiolabelled antibody to target tumours for imaging or detection. It is aimed at more specific and sensitive tumour imaging. A glycoprotein TAG 72 has been found to be useful target antigen in colorectal cancer, the expression of which has been found to be high as 94% in colonic adenocarcinoma.

A study using indium III labeled TAG 72 for imaging colon cancer showed sensitivity of 70%. Specificity -90% and accuracy 72%. Radioimmuno guided surgery (RIGS) utilizes Radiolabelled antibody to localize tumour. Detection achieved by a hand held gamma camera detecting problems during surgery.

BIOPSY:

HPE of the specimen obtained preoperatively either by open or via colonoscopy or Proctosigmoidoscopy or laparoscopy is done to study the type, grade of tumour and other characteristics [29]

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Running cancer detection clinics where patients over 45 years are investigated thoroughly for visceral malignancies, every year is out of question for our rural and urban population. Public education regarding early signs of lower GI Malignancies are also not met with much success.

Most of the cases of carcinoma rectum can be detected by a proper rectal examination and this should be mandatory for any patient presenting with suggestive symptoms or a history of piles. Colonic cancers need further investigations described earlier. Faecal occult blood should be done for every patient at high risk. Patients at high risk should be screened by luminal contrast studies.

The investigations available in this hospital apart from routine basic investigations are luminal contrast studies, colonoscopy, USG abdomen and CT scan. In patients, who presented as acute emergencies, diagnosis was confirmed by exploratory laparotomy.

DIFFERENTIAL DIAGNOSIS:

Conditions simulating colonic and rectal carcinoma include ileocaecal tuberculosis, appendicular mass, diverticulitis, polyps, Crohn's Disease, Ulcerative colitis etc. Rectal carcinoma should be differentiated from squamous cell carcinoma of anal canal as the treatment is entirely different for this.

TREATMENT

The various modalities of treatment for Lower GI Malignancies are:

1. Surgery
2. Radiotherapy
3. Chemotherapy

SURGERY:

Surgical resection remains the mainstay of treatment and core of Lower GI Malignancies. Due to biological unfavourable response to chemotherapeutic drugs and radiotherapy due to various reasons, surgery is still the best palliative treatment modality [4].

The objective of surgery in treatment of lower GI malignancies is to remove cancerous segment of bowel, the mesentery containing its lymphatics, lymph nodes and any organ directly invaded by the tumour. The way surgical resection is done consists of major steps to give therapeutic curative resection with as little morbidity and mortality to the patient and starts preoperatively itself.

Preoperative bowel preparations:

In this study, a single preoperative Peglec preparation was given the day before surgery starting around 11.00 am and ending around 5.00 pm encouraging the patient to take clear fluids till 10.00 pm. Supportive

IV fluids were used then and there. Complete luminal obstruction was an absolute contraindication for Peglec preparation. No luminal antibiotics were used. Only preoperative antibiotics were used at the induction time.

Surgical Technique:

Techniques to minimize tumour spillage, remove adequate bowel length and bowel continuity restorations are as important as surgery itself. The Abdomen was opened & tumour was assessed for resectability by :

Palpating liver for secondaries by visual, palpatory and intraoperative USG if available. (Isolated / 3 metastases within one resectable lobe is not a contraindication for curative surgery).

- Palpate peritoneum, draining lymph nodes and mobility and fixity of tumour to adjacent organs or abdominal wall structures.

Anatomical resection of lower GI malignancies at different sites

Tumour Location	***Vascular Ligation***	***Bowel Resection***
Jejunum and ileum	Jejunal & Ileal Branches	With adequate margins
Caecum, ascending colon	Ileocolic, Right Colic	Right hemicolectomy
Hepatic flexure , Proximal transverse colon	Ileocolic, Right Colic, Middle Colic	Extended right hemicolectomy with omentectomy

Distal transverse colon splenic flexure	right colic, middle colic, left colic	Extended right hemicolectomy with omentectomy or left hemicolectomy
Descending colon	Inferior mesenteric or left colic	Left hemicolectomy
Sigmoid colon	Inf. Mesenteric or sigmoid	Left hemicolectomy or sigmoid resection
Rectum	Inf. Mesenteric or Rectal	A.P Resection / anterior resection

Then intestine to be resected is tied both proximally and distally to prevent intraluminal spread [6]. The major segmental artery supplying the cancerous bowel is ligated, and divided. Any adhesion to adjacent structures are resected in toto if feasible to avoid loss of curative resection (most adhesion, lymphadenopathy may be inflammatory and can only be proved histologically).

The extent of resection depends upon primary location of the tumour, lymphatic metastases, adjacent organ invasion. To get a tumour free margin, usually 2.5 cm margin clearance is ideal, and anastomosis may be end to end or end to side which should be tension free.

TREATMENT OPTIONS DEPENDING ON TUMOUR LOCATION

Jejunal / ileal tumours:

Treated by curative resection with adequate margins. (Fig.15)

Carcinoma Caecum or Ascending Colon :

Treated by a Right Hemicolectomy . (Fig.16)

Carcinoma Hepatic Flexure:

Treated usually by Right hemicolectomy or extended right hemicolectomy. (Fig. 17, 18).

Carcinoma Transverse Colon :

Depending on position of tumour on Transverse colon. Segmental excision of transverse colon. Extended right hemicolectomy may be performed.

Carcinoma Splenic Flexure:

Treated by resection of colon including transverse, descending if needed sigmoid colon with associated lymphatics and mesentery., Left Hemicolectomy. (Fig. 19,20).

Summarizing the various treatment modalities for rectal carcinoma is as follows [46]:

(Extra peritoneal rectum extending upto 11 cm from anal verge)

- ❖ Abdomino perineal resection (Fig.21,22)
- ❖ Low anterior resection:
    - End to end / side to end

- Sutures / Staples

❖ Abdomino sacral resection

❖ Coloanal resection

- Endoanal / Pull through
- Staples / Sutures
- End / J.Pouch

❖ Localised procedures

- Local excision
- Fulguration
- Endocavity irradiation or brachy therapy

Tumour free margins are usually achievable for colon cancer and usually a margin of 5 cm is deemed adequate, since <2.5% of tumour have intramucosal spread more than 2 cms[36]. During low anterior resection, distal margin of 2 cm is deemed adequate for well differentiated, small non bulky tumour with favourable prognostic features thereby aiding restoration of intestinal continuity 8.

When synchronous colonic cancers are found at different sites in the colon, the preferred procedure is subtotal colectomy. Resection of contiguous organs for locally invasive tumours in approximately 10% of cases[37]. Most commonly involved organs are bladder, ovary, ureter, abdominal wall, less frequently small intestine, spleen, pancreas, stomach and uterus [39].

SURGICAL INTERVENTION IN EMERGENCY PRESENTATION:

The major emergency presentation is obstruction, and perforation with later occurring 3 – 8% of all cases and occurring combination in about 2% of cancer cases [5].

Obstruction:

Immediate surgical treatment is necessary. The surgery is planned and carried out. Obstructing cancer either in small bowel or right / transverse colon is treated by Resection and primary anastamosis with adequate margins, while left sided cancer obstruction is more a difficult problem [50]. Appropriate surgical procedure depends on location, findings, surgeons' experience and judgement. It can be treated in stages with defunctioning colostomy and later curative surgical resection with anastamosis.

Various modalities available:

- Primary resection with exterioration of both ends.
- Hartmann's procedure
- Primary resection with anastamosis
- Primary resection with anastamosis protected by proximal colostomy or ileostomy.
- Subtotal colectomy with ileosigmoidostomy

PERFORATION

It is usually a life threatening emergency. Thorough exploration of peritoneal cavity is mandatory. Sometimes it may be difficult to distinguish mass caused by diverticulosis, perforation from a perforated colonic cancer[40].

Goal of surgery [45]:

To remove diseased, perforated segment as dehiscence rate is very high in a contaminated field. To do anastamosis protected by proximal colostomy / ileostomy. The diverting stoma closure is done 10 weeks later, the peritoneal contamination thoroughly irrigated and sucked out. The survival rate reaches upto 30% of curative resection at 5 yrs in these patients [41].

LAPAROSCOPIC SURGICAL INTERVENTION:

Usually laparoscopic assisted methods are used. Fully laparoscopic methods are also attempted. They are claimed to facilitate patient convalescence, reduce stress with better post op outcome [17,18].

Adjuvant Treatment for lower GI Malignancies:

Chemo radiation forms the main stay of treatment for anal canal Tumours [47].

Radiotherapy:

The role of RT is well defined in rectal carcinoma than colonic and small bowel tumours[35]. This is due to variation in local presentation after

surgery. In colonic carcinoma the failure site is abdominal rather than local site which makes radiotherapy less feasible [26]. In rectal cancer local failure with recurrence is the most common presentation making adjuvant radiotherapy worth while[34]. In rectal carcinoma, local recurrence in pelvis is due to inability to do wide resection due to narrow confines of pelvis which is now overcome by laparoscopic methods. Radiotherapy can be used in various regimens to prevent local failure [23].

- ❖ Can be used preoperatively
- ❖ Sandwich technique
- ❖ Post operative irradiation.

Usually 4500- 5000 cGy is delivered over 5 – 7 week period and little complications results during preoperative use and complications are more after post operative irradiation.

Unresectable tumours, patients refusing surgery may be treated with palliative radiotherapy. Also can be used for palliation of painful recurrences and bony metastases. Radiotherapy can be applied using Brachy therapy and Teletherapy.(Fig.25,26). Most commonly used teletherapy are Cobalt therapy and Neutron beam irradiation. (Fig.27) Radioactive isotopes are used in brachy therapy with short half life.

CHEMOTHERAPY:

Adeno carcinomas of lower GI tract have usually been resistant to chemotherapeutic agents. But CT is appealing and most effective when

tumour burden is minimal / smallest and when the fraction of malignant cells in growth phase is highest. It is most commonly given following surgical procedures.

Hence maximum response is obtained for postoperative regimen usually for stage III disease with no significant improvement in patient with stage II disease. Most experimented drugs are 5 FU alone or 5 FU and Cyclophosphamide.,or 5-FU with Levamisole combination with leucoverin [51] . Other Drugs include vincristin , irinotecan, oxaliplatin [14].,

Currently systematic CT is used to improve long term survival by reducing incidence of distant metastases, to improve local disease control, by combination with external beam radiation, found to have modest survival benefits in stage II & III patients. The most promising drug, 5FU, which acts as radio sensitizer in the chemoradiation techniques [19].

Immunotherapy:

Various agents have been tried to modulate or stimulate innate immune response or enhancement of immune response to destroy, prevent tumour progression, implantation or metastasis. No encouraging results are reported after randomized trials. Commonly used agents are levamisole, BCG vaccination , autologous vaccine (irradiated autologous tumour cells with BCG)

Another strategy is passive – specific immunotherapy using monoclonal antibodies especially against CEA antigen and various other

tumour antigens, which is said to result in improved disease free, and overall survival. Of recent interest is MCA 17 – 1A, which resulted in some improvement in Duke Stage C patients. Also MCA – attached with tumoricidal drugs used to target metastases, local recurrences etc., all are still in experimental stages.

Treatment of metastatic disease:

Hepatic metastasis:

It is important to biopsy liver metastasis for histological diagnosis. Patients with upto 2 or 3 liver mets confined to one lobe of the liver may be offered liver resection.Multiple painful hepatic metastasis can be palliated by cytotoxic drugs, cryotherapy and laser therapy.

Follow up of lower GI malignancies:

The objective of follow up is for early detection of recurrence or a metachronous carcinoma that can be treated surgically. The follow up program in general consists of periodic history and physical examination, fecal occult blood (FOB), CBC, liver function tests, tumour markers, colonoscopy and radiographic studies. Colonoscopy is an important component of the program. It is a safeguard for detecting anastamotic recurrence, missed synchronous lesions and metachronous tumours at early stages[38]. In this study a standard questionnaire was used for all patients in the follow up period also.

MATERIALS AND METHODS

- This study consists of patients admitted for the management of lower GI malignancies in Surgical Wards of Coimbatore Medical College Hospital, Coimbatore. Period of Study – 2006 to 2008
- On admission all the patients were subjected to basic blood investigations, Test for fecal occult blood, USG Abdomen, Barium studies, CT abdomen and UGI scopy.
- Following clinical and proctoscopic examination, Colonoscopy was done for all patients – except for emergency cases and biopsies were taken.
- Bowel preparation was done preoperatively using plain oral fluids, and PEG preparation – the day before surgery.
- Per operative findings regarding peritoneal implants, liver secondaries, peritoneal implants and tumour invasion to the nearby structures were noted.
- Post operative course was closely observed until the first follow up visit.
- Patients with permanent colostomy were followed up for colostomy care.

OBSERVATIONS AND DISCUSSION

In this series of 78 cases of lower GI malignancies the following observations were made.

Risk Factors:

Diet:

Only 4 of the 78 patients were pure vegetarians. The majority of patients were illiterate, they could not specify the exact dietary constituents. But most patients gave history of consumption of fat and spicy food, fibre intake was moderately adequate in most patients.

Tobacco:

56 patients out of 78 were using tobacco in some form or other. All the male patients except 5 were regular smokers of beedi or cigarettes. 26 out of 33 female patients were using tobacco in the form of tobacco, pan masala etc [41].

Incidence:

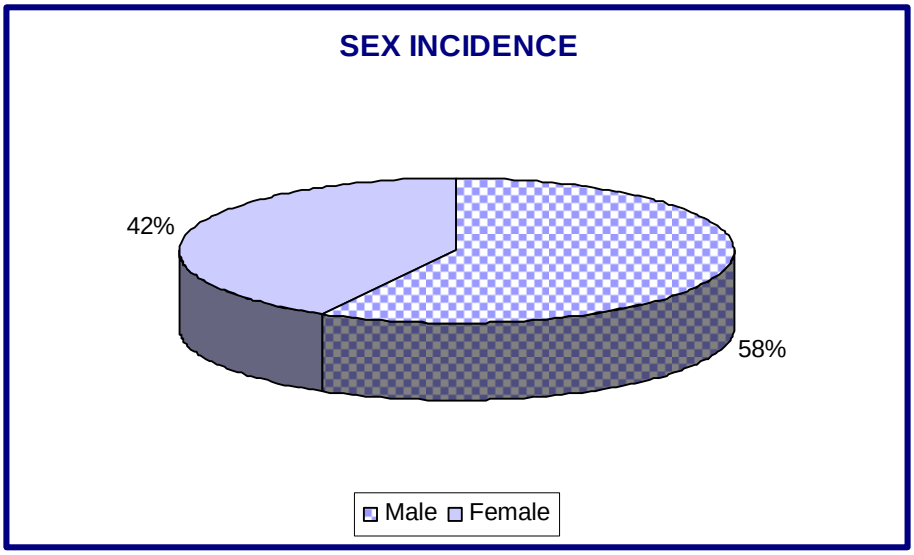
Age :

Most of the cases were from older age group. Maximum incidence was in the sixth decade i.e. 29 cases in the age group 51 – 60 yrs [14].

SEX INCIDENCE

Male	Female
45	**33**

M : F = 45:33 = 1.36:1

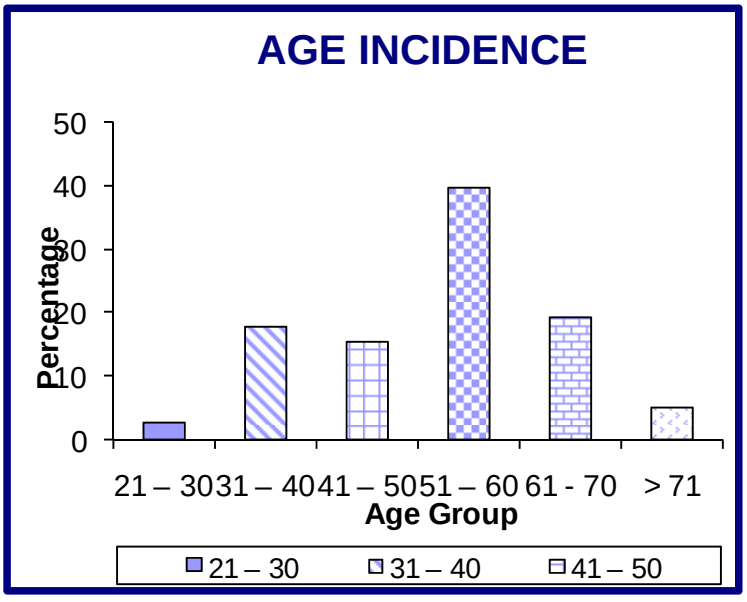


Sex:

Incidence was relatively more in males. M:F ratio in this series was 1.36:1. Incidence was nearly equal in rectal carcinoma with slight male preponderance in other site carcinomas [4].

AGE INCIDENCE

Range	Male	Female	Total	Percentage
21 – 30	1	1	2	2.56
31 – 40	7	7	14	17.94
41 – 50	6	6	12	15.38
51 – 60	17	14	31	39.74
61 – 70	10	5	15	19.23
> 71	4	0	4	5.12
Total	**45**	**33**	**78**	**100**



Site:

Carcinoma rectum accounted for majority of cases. In 78 patients, 33 were carcinoma of rectum (42.30 %). The distribution of tumour in the other sites in the decreasing order of frequency were as follows., Small intestine 4, Colon 27, Rectum including Rectosigmoid junction 33, anal canal 14, each.

48 out of 78 were left sided lesions.

Mode of Presentation:

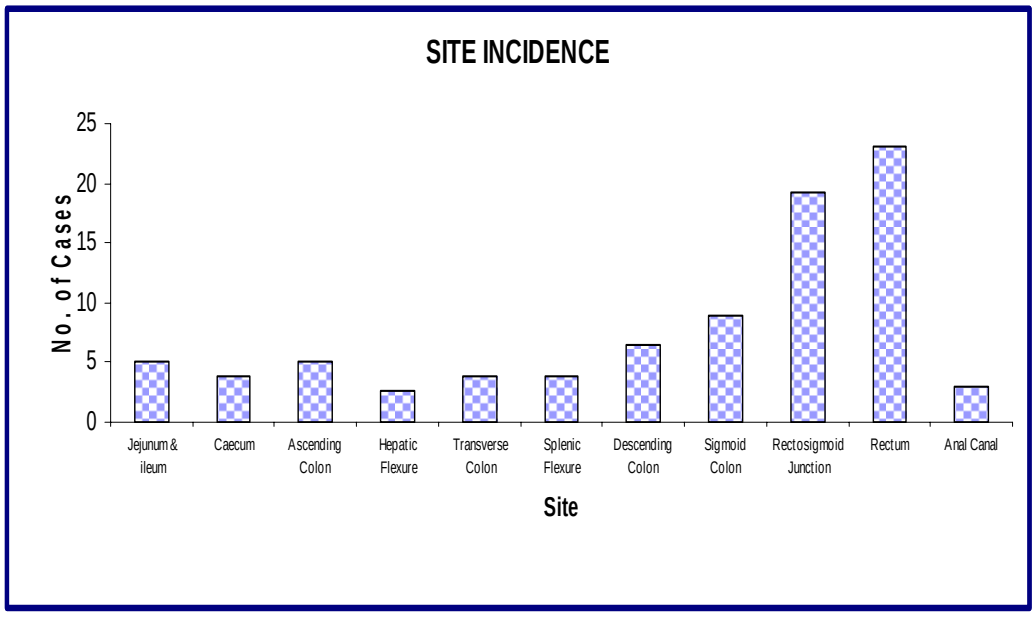
Majority of the patients though had symptoms for sometime, tend to ignore them and presented at late stages.

17 of the cases in this series presented as acute emergencies (21.79%). 12 of them presented with obstructive features. Out of which 3 were acute on chronic bowel obstruction. 5 patients presented with features of bowel perforation with peritonitis [50].

Majority of the cases of right sided colonic tumours had symptoms of altered bowel habits, increasing constipation, bleeding per rectum, being the major complaint. Some patients presented with spurious diarrhea. Majority of bleeding per rectum were seen in rectum and sigmoid lesions .Pain was relatively late symptom. Abdominal lump was presented in 5 cases of rectosigmoid growth.

SITE INCIDENCE

Site	Male	Female	Total	Percentage
Right Sided Tumour	9	7	16	20.51
Left Sided Tumour	37	11	48	61.53
Anal Canal	8	6	14	17.94



SITE INCIDENCE

Site	Male	Female	Total	Percentage
Jejunum & ileum	3	1	4	5.12
Caecum	2	1	3	3.84
Ascending Colon	2	2	4	5.12
Hepatic Flexure	1	1	2	2.56
Transverse Colon	1	2	3	3.84
Splenic Flexure	1	2	3	3.84
Descending Colon	2	3	5	6.41
Sigmoid Colon	5	2	7	8.97
Rectosigmoid Junction	9	6	15	19.23
Rectum	11	7	18	23.07
Anal Canal	8	6	14	17.94
Total	**45**	**33**	**78**	**100**

LOWER GI MALIGNANCIES

SIGN AND SYMPTOMS IN THE SERIES

No. of Cases	Jejunum & Ileum	A. Colon	D. Colon	T. Colon	Sigmoid Colon	Rectum/ Anal Canal
No. of Cases	4	9	8	3	7	47
Signs / Symptoms						
Pain	2	8	11	3	4	10
Altered Bowel habits	1	4	8	1	3	12
Bleeding per rectum	-	5	4	-	-	18
Mucus per rectum	-	-	10	-	2	16
Tenesmus	-	-	8	2	3	12
Lump Abdomen	-	-	-	-	3	2
Hepatomegaly	-	4	-	-	3	3
Weight Loss	2	10	20	3	2	18
Lassitude	1	10	20	4	4	18
Anemia	1	10	18	2	3	16
Acute Obstruction	1	-	3	-	2	6
Peritonitis	-	-	1	1	1	2

Rectal growth was palpated in most cases of carcinoma rectum on per rectal examination.

Transverse colon growth presented with pain abdomen with typical history of diarrhoea alternating with constipation. Anemia, anorexia and progressive loss of weight was present in majority of cases at all sites.

12 patients presented with symptoms of metastasis / disseminated disease. 3 presented with skeletal metastases mainly in lumbo dorsal spine ., out of which one presented with paraplegia. 10 cases presented with hepatomegaly, one patient presented with cough, haemoptysis, with multiple lung metastases.

Diagnosis:

In this series, diagnosing cases of carcinoma of rectum & anal canal was not a problem due to late presentation in most of the cases. One case of left colon growth perforation was missed due to extensive peritonitis with adhesion treated with conservative management. Right sided growth tend to present as mass and left sided growth with features of mass or obstruction & most of rectal growths were palpated except for a few. Proctoscopic examination was done followed by tests for fecal occult blood. USG Abdomen & CT Abdomen was done for all cases & initial phase of the disease was studied. Colonoscopy was done for all cases, except emergency cases & biopsies taken from the lesions under vision or from suspected areas. Contrast studies with Barium ,i.e., Barium

meal or Barium enema was done. CEA levels were done for 3 cases., levels were raised in all three cases ., average 39 ng / ml .

Pathology and Stage:

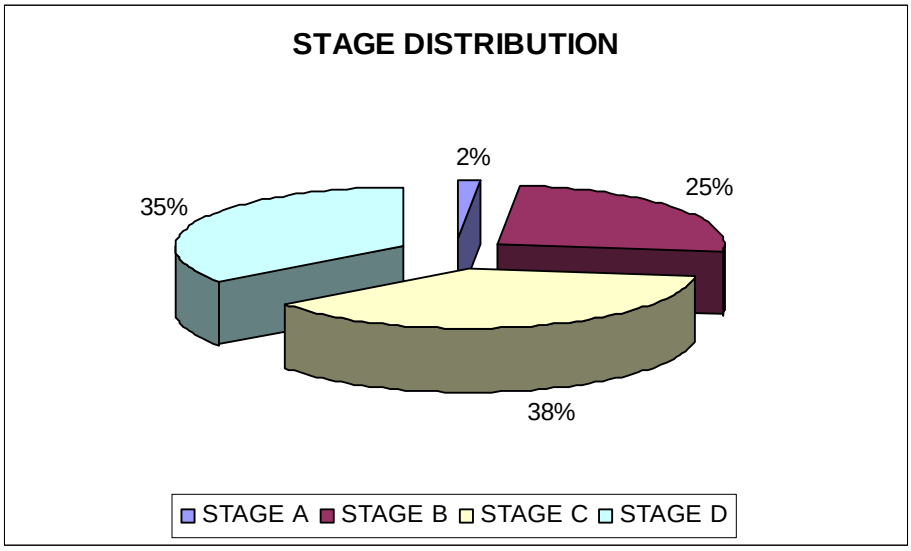
Most of the rectosigmoid and left sided growths were either annular and stenosing or ulcerative with infiltration type. Almost all cases of carcinoma rectum were of ulcerative type. 3 sigmoid growth showed infiltration into surrounding viscera. 2 cases of carcinoma rectum presented with posterior vaginal wall infiltration. One patient presented with B/L hydronephrosis due to ureteric compression.

HISTOPATHOLOGY:

Sl.No.	Types of CA	No. of Cases
1.	Adeno Carcinoma	64
2.	Squamous Carcinoma (Anal Canal)	12
3.	Malignant Melanoma (Anal Canal)	2

STAGING – COLON & RECTUM

Stage	Male	Female	Total	Percentage
A	0	1	1	1.67
B	8	7	15	25
C	10	13	23	38.33
D	14	7	21	35
Total	**32**	**28**	**60**	**100**



Majority of right sided growths were polypoidal or cauliflower like growths. 1 hepatic flexure growth was found to infiltrate into abdominal wall. 1 case of multiple polyposis coli with rectal adenocarcinoma infiltrating into bladder was diagnosed [25]. 2 cases of synchronous tumour found, one was synchronous rectal carcinoma and transverse colon growth. While the other was synchronous gastric carcinoma (antrum) and colonic carcinoma.

STAGING: (COLON & RECTUM)

Only 1 case presented in Duke Stage A (1.67%), 15 were in Stage B (25%), 23 were Stage C (38.33%), 21 in Stage D (35 %). Out of 12 disseminated malignancies 10 had multiple liver metastases. While two had dorsolumbar spine metastasis. One patient presented with multiple lung secondaries. Two presented with peritoneal metastasis in the form of nodules.

Grading:

Moderately differentiated tumour predominated. 41 out of 78 cases belonged to this (52.56 %), 21 cases were well differentiated (26.92%) and 14 cases were poorly differentiated (17.94 %) type.

Treatment Adopted:

Surgical resection in the form of either curative or palliative resection or palliative bypass / colostomy was attempted on all patients except 10 . In these 10 , 4 refused surgery and were treated with CT/RT while remaining went against medical advice.

Emergency Cases:

17 patients presenting as emergencies were taken up., 5 patients had perforation with peritonitis. One case of descending colon, 3 cases rectosigmoid junction growth and one carcinoma rectum.

The first has presented with perforation near the proximal margin of growth treated with transverse loop colostomy and underwent elective resection and anastomosis. One rectal carcinoma presented with caecal perforation treated with ileostomy and elective APR later.

12 cases presenting with bowel obstruction were taken up for emergency surgery [20]. Out of 6 rectal growths 3 were inoperable, treated with Hartmann procedure with end colostomy. (Fig.29,30). Remaining were treated with temporary defunctioning colostomy with elective APR later.(Fig.31,32). Out of 3 growths in rectosigmoid / sigmoid colon, 2 were inoperable with extensive local infiltration, treated by transverse loop colostomy. While in one resection (palliative) done with Hartmann's procedure. One caecal growth was inoperable which was treated by ileotransverse anastamosis.(Fig.16)

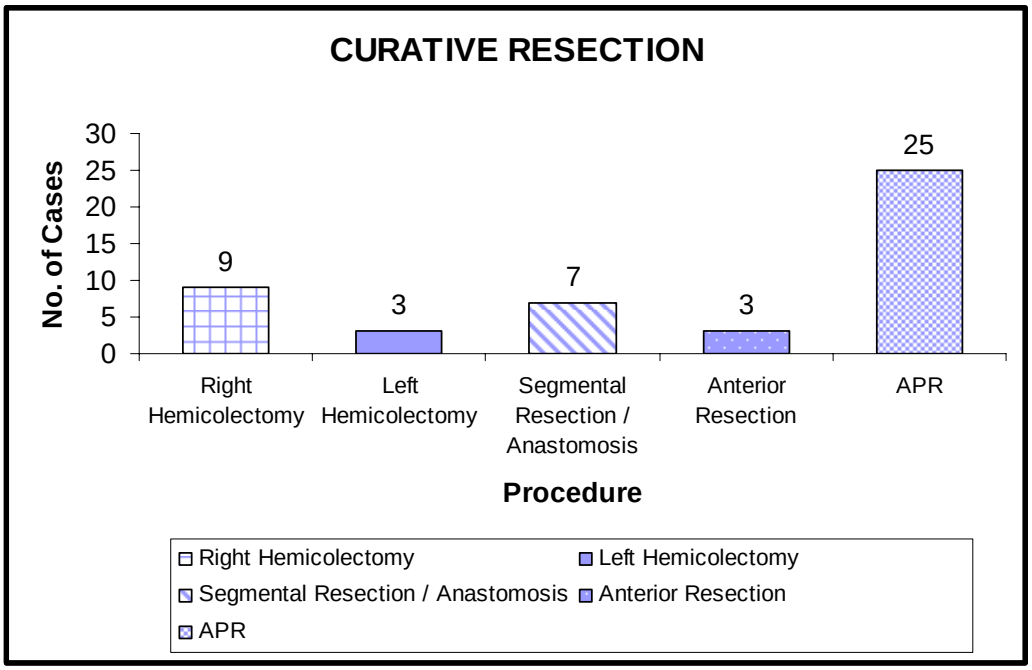
Elective Cases:

51 patients were treated with elective surgery in whom 25 rectal carcinomas underwent curative APR with 3 cases treated with anterior resection with colo anal anastamosis [27].(Fig.21,22). 4 cases underwent Haartmann's procedure. 3 left colon growth were treated with left hemicolectomy (Fig.19,20) and 9 patients underwent right hemicolectomy (Fig.17,18)., with 2 extended hemicolectomies for transverse colon growth, other included cancer caecum and ascending colon. Circular staplers were used for 4 cases and sphincter saving surgery was performed. (Fig.28).

Sl.No.	Surgery	Numbers
1.	Elective Cases	51
2.	Emergency Cases	17

CURATIVE RESECTION

Right Hemicolectomy	9
Left Hemicolectomy	3
Segmental Resection / Anastomosis	7
Anterior Resection	3
APR	25



Segmental resections were done in 7 cases which, were for jejunoileal growth.,rectosigmoid growth & transverse colon growth.

ADJUVANT THERAPY:

The following Regimen was used:

I. CA COLON :

Inj.Oxaliplatin 100 mg / m^2 from D_1 to D_3

Inj. 5 FU 750 mg from D_1 to D_3

Inj. Leucoverin 30 mg from D_1 to D_3

The above drugs were given for six cycles.

II. CA RECTUM:

The above drugs were given for two cycles followed by radiotherapy and then 2 more cycles of chemotherapy.

III. CA ANAL CANAL:

Inj. Mitomycin C 12 mg on D_1

Inj. 5 FU 750 mg on D_1 to D_4. With concurrent RT (6000 cGy @ 200 cGy / day).

48 cases received adjuvant chemotherapy. 8 people refused further adjuvant therapy. Radiotherapy was given preoperatively to one patient and later underwent Haartmann's procedure for (carcinoma rectum) obstruction.

Complications:

Significant post operative complications included anastamotic leak, faecal fistulae occurred in 7 patients. Surgical site infection occurred in 9 patients which were treated accordingly. General complications like post operative respiratory infection, uraemia, electrolyte imbalance were found in 12 patients. Most of them belonged to older age group with associated diabetes mellitus and poor general status. One case of stapler anastomosis had leak which needed revision.

Follow up:

Follow up was average with most patients failing to turn up after 3 – 6 cycles of chemotherapy, 10 patients attended the clinic for a long time in the follow up period in this study on an average of 20 months and 1 patient was followed up for 28 months which was the longest. 8 patients developed multiple hepatic metastases during follow up. While 3 patients had local recurrence, 18 patients had regular follow up with repeat USG and basic blood investigations being normal.

CONCLUSION

- Most patients were non vegetarians taking a high fat diet. But no definite evidence of diet as a risk factor could be identified.
- Commonest age group of Lower GI Malignancies was on the 6^{th} decade.
- Incidence was higher in males, M : F was 1.36 : 1
- Carcinoma Rectum was the most commonest site of large bowel (42.30%) in the series.
- 17 cases presented with acute emergency in the form of bowel obstruction or perforation.(21.79 %)
- Use of Staplers have enabled Sphincter saving surgeries to be done & avoids a Permanent Colostomy.
- To conclude, any patient above 40 years of age, when complaints of altered bowel habits or bleeding per rectum, every effort should be made thoroughly to investigate him to find out if he harbors malignancy of lower GI tract.

CASE STUDY PROFORMA

Name : Age / Sex :

IP No. : DOA :

Presenting Complaints

:

1.	Bleeding per rectum		
2.	Abdominal Pain		
3.	Tenesmus		
4.	Early morning spurious diarrhoea		
5.	Constipation		
6.	Altered bowel habits		
7.	Features of intestinal obstruction		
8.	Loss of weight		
9.	Loss of appetite		
10.	Hemetemesis, Malena		
11.	Mass descending per rectum		
12.	Back pain		
13.	Urinary symptoms		
14.	Features of Carcinoid syndrome (Flushing, diarrhoea, cyanosis)		

Previous History of any surgeries for : …….

General Condition of the patient : …….

P.R. :

B.P. :

Blood Grouping & Typing :

Clinical Findings :

Per Rectal Examination :

Growth Position :

Investigations :

1. Urine Albumin :

Sugar :

Deposits :

2. Blood Sugar :

Urea :

Serum Creatinine :

Complete Hemogram : Hb% :

TC : DC :

LFT : ESR : Platelets :

Chest X- RAY : ECG :

X – RAY Abdomen :

Motion for Occult blood :

Barium Contrast Studies : Barium Enema

Barium Meal

CT Abdomen : CECT :

Colonoscopy :

HPE Grading :

INTERVENTION :

Surgical

Excision

OPERABLE

Resection and anastamosis :

Anterior Resection :

Abdomino Perineal Resection :

INOPERABLE :

PREOPERATIVE FINDINGS

Local :

Lymph Nodes :

Peritoneal Implants :

Liver Secondaries :

POST OP FOLLOW UP :

Adjuvant Therapy :

QUESTIONNAIRE

(Model)

Name : Mr.Venkatasamy

Age / Sex : 72 / M OP / IP No. : 10011

Address : P.N. Palayam, Coimbatore.

1.	Do you have abdominal pain?	Yes / ~~No~~
2.	Where did the pain first start?	Lower Abdomen
3.	Character of pain. Pricking □ Dull Aching ✓ Colicky □	
4.	Relation with food.	~~Yes~~ / No
5.	Relation to defecation.	Yes / ~~No~~
6.	Radiation of pain.	~~Yes~~ / No
7.	Do you have any night pain?	~~Yes~~ / No
8.	Do you have painful incomplete defecation association with bleeding?	Yes / ~~No~~
9.	Do you have bleeding per rectum?	Yes / ~~No~~
10.	Amount of bleeding:	
	Mild	-
	Moderate	✓
	Severe	-
11.	Is the bleeding associated with pain?	Yes / ~~No~~

12.	Do you have constipation on and off?	Yes / ~~No~~
13.	Do you have any change in bowel habits recently?	Yes / ~~No~~
14.	Do you have early morning spurious diarrhea?	Yes / ~~No~~
15.	Do you have fecal incontinence?	~~Yes~~ / No
16.	Do you have passage of mucus and blood while attempting at defecation?	Yes / ~~No~~
17.	Do you have a feeling of any mass descending per rectum?	~~Yes~~ / No
18.	Do you have perianal pruritus?	~~Yes~~ / No
19.	Do you have loss of weight?	Yes / ~~No~~
20.	Do you have loss of appetite?	Yes / ~~No~~
21.	Do you have abdominal distension?	~~Yes~~ / No
22.	Do you smoke?	Yes / ~~No~~
23.	Are you an alcoholic?	~~Yes~~ / No
24.	Do you take mixed diet?	Yes / ~~No~~
25.	Do you take any drug for a prolonged period?	~~Yes~~ / No
26.	Have you undergone any surgery before?	~~Yes~~ / No
27.	Is any other family member suffers / suffered from similar complaints?	~~Yes~~ / No

BIBLIOGRAPHY

1) Short Practice of Surgery. Bailey & Love 24th Edition (P. 1153 - 1272)
2) Maingot's Abdominal Operations 11th Edition (P. 479 to 757)
3) Sabiston Textbook of Surgery 18th Edition Vol. 2 (P.1219 to 1241 & 1323 - 1573)
4) Schwartz's Principles of Surgery 8th Edition. (P. 1017 to 1137)
5) Hamilton Bailey's Emergency Surgery 13th Edition (P. 393 - 446)
6) Mastery of Surgery Fourth Edition, Rober J.Baker, Joseph E.Fischer, (P. 1399 - 1689)
7) Principles of Oncology – Devita Hellman, Rosenberg (P. 1036 - 1135)
8) Oxford Textbook of Surgery. 2nd Edition (P. 1345 - 1611)
9) Principles and Management of Cancer – A Practical Guide (Ranajit Sen) (P. 154 - 157)
10) UCSF Medical Centre TNM Staging of Colon & Rectum
11) Recent Advances in Surgery (26) 2003 (Wendy Atkin, John Marshover) (P. 111 - 120)
12) Recent Advances in Surgery for Rectal Cancer (9) 2004. PJ Shukla, HA Kanhex SV Shrikand (P. 263 - 272)

13) Sphincter Saving Surgery for Cancer Lower Rectum with Special Emphasis on Abdominosacral Resection (Recent Advances in Surgery - 9) Dhananjaya Sharma (P. 275 – 282)

14) Current Surgical Diagnosis and Treatment – 12th Edition. 2006 (P. 652 - 763)

15) Gastroenterology Clinics of North America - 2007 (Colorectal Cancer in IBD AIN & other Neoplastic Precursor lesions of the Anal Canal and Perianal region)

16) Randomised Clinical trial of effect of Synbiotics, neomycin & mechanical bowel preparation on intestinal barrier function in patients undergoing colectomy. – British Journal of Surgery 2007-94 (546 - 554)

17) Local Excision of rectal tumours by transanal endoscopic microsurgery – British Journal of Surgery 2007 – 94 (627 - 633)

18) Early introduction of laparoscopic sigmoid colectomy during residency. – British Journal of Surgery 2007 – 94 (P. 634 - 641)

19) Colorectal Cancer – Clinical Review (Anne B Ballinger, Clive Angiansah) (P.715 - 718) British Medical Journal Oct 2007 Vol. 335

20) Signet ring cell Ca of Appendix presenting with intestinal Obstruction. – Indian Journal of Surgery Feb 2007 Vol. 69 (P. 22-24)

21) Optical Adjuncts for Enhanced Colonoscopic Diagnosis. – British Journal of Surgery 2007; 94 (P. 6 - 16)

22) Role of Circulating tumour cells in predicting recurrence after excision of primary colorectal carcinoma – British Journal of Surgery 2007. 94 (P. 96 - 105)

23) Impact of Radiotherapy on local recurrence of rectal cancer in Norway. – British Journal of Surgery 2007 – 94 (P. 113 - 118)

24) Multicentric Randomised clinical trial of mechanical bowel preparation in elective colonic resection. British Journal of Surgery 2007; 94 (P. 689 - 695)

25) Young Age Colorectal Cancer and Identification of Hereditary Non – Polyposis Colorectal Cancer Cohorts. – British Journal of Surgery 2007; 94 (P. 1055 - 1056)

26) Size of Surgical Margin does not influence recurrence rates after curative liver resection for colorectal cancer liver metastases – British Journal of Surgery 2007. 94 (P. 1133 - 1138)

27) Oncological outcome of Ultra low coloanal anastomosis with and without intersphincteric resection for low rectal adenocarcinoma – British Journal of Surgery 2007. 94 (341 - 345)

28) Leptin acts as a mitogenic and antiapoptotic factor for Colonic cancer cells. – British Journal of Surgery 2007. 94 (P. 346 - 354)

29) Clinical risk score predicts yield of staging laparoscopy in patients with colorectal liver metastases. – British Journal of Surgery 2007.94 (P. 855 – 859

30) Role of Ultrasonography in the detection of unresectable recurrence after lobectomy for colorectal liver metastases. – British Journal of Surgery 2007. 94 (P. 1403 - 1407)

31) Population based study of local surgery for rectal cancer – British Journal of Surgery 2007. 94 (P. 1421 - 1426)

32) Prediction of Lateral Pelvic node involvement in low rectal cancer by conventional Computed tomography. – British Journal of Surgery 2007. 94 (P. 1014 - 1019)

33) Immuno Histochemical Staining for Mismatch repair proteins and its reliance in the diagnosis of HNPCC – British Journal of Surgery 2007.94 (P. 1020 - 1027)

34) Preoperative but not postoperative systemic Inflammatory response correlates with survival in colorectal cancer. – British Journal of Surgery 2007.94 (P. 1028 - 1032)

35) Inspired Supplemental Oxygen reduces markers of oxidative stress during elective colon surgery - – British Journal of Surgery 2007.94 (P. 475 - 477)

36) A Protocol in not enough to implement an enhanced recovery programme for colorectal resection – British Journal of Surgery 2007; 94 (P. 224 - 231)

37) Extended abdomino perineal resection with gluteus maximus flap reconstruction of the pelvic floor for rectal cancer. – British Journal of Surgery 2007.94 (P. 232 - 238)

38) Clinicopathological significance of microscopic abscess formation at the invasive margin of advanced low rectal cancer - – British Journal of Surgery 2007 (P. 239 - 243)

39) Impact of surgeon volume and specialization on short term outcomes in colorectal cancer surgery – British Journal of Surgery 2007.94 (P. 880 - 889)

40) Differences in Patient post operative and long term outcomes between obstructive and perforated colonic cancer. (Dept. of Surgery, Colorectal Unit, University of Barcelona, Spain) Pub Med.

41) Quality of Life Assessments in Colorectal Surgery (Jess.P. Hillaned Hospital, Kirurgisk Klinik Pub Med.)

42) Pathologic Basis of Disease – Robbins 7th Edition. P 828 – 875

43) Principles of Internal Medicine – Harrison – 16th Edition (P. 1763 - 1808)

44) A.S.I. Text Book of Surgery 2003 Edition (P. 431 - 523)

45) Essential Surgical Practice by Cushierie (Third Edition) (P. 1297 - 1408)

46) Atlas of Operative Surgery – Zollingher (P. 363 - 478)

47) American College of Surgeons Textbook of Surgery(Principles & Practice) 2005 Edition (P. 403 - 755)

48) Textbook of Radiology & Imaging. Vol 1. David Sufton 7th Edition. (P. 615 - 691)

49) Clinicopathological characteristics of colonic carcinoma in relation to localization and histologic type. (Article in Serbian – Pub Med PMID 18 35 79 06)

50) Results of Surgery for Colorectal Carcinoma with obstruction – Surgical Department, Didmotichon Gen. Hospital, Greece. Pub Med PMID : 18299883

51) Second line chemotherapy use in metastatic colon cancer varies by disease responsiveness. (University of Texas Health Science Center, Honston, TX, USA) Pub Med PMID : 18279578

MASTER CHART										
Sl.No.	**Name**	**Age**	**Sex**	**IP. No**	**Diagnosis**	**Type of Growth**	**HPE**	**Type of Resection**	**Emergency / Elective**	**CT / RT**
1.	Mangalam	70	F	23824	CA Rectum	ULP	PDA	APR	ELE	-
2.	Nagalakshmi	50	F	42247	Splenic Flexure Growth	PAP	WDA	SRA	ELE	ADJ
3.	Ushakumari	61	F	58220	CA Anal Canal	FUG	MM	-	-	CRT
4.	Palaniappan	70	M	31042	CA Rectum	INF	PDA	APR	ELE	ADJ
5.	Madheswaran	55	M	33710	CA Rectum	PAP	WDA	APR	ELE	ADJ
6.	Chinnasamy	70	M	33936	CA Ascending Colon	ULP	WDA	RHC	ELE	-
7.	Selvarani	56	F	36812	CA Anal Canal	ULP	PDS	-	-	CRT
8.	Chinthamani	50	M	38416	CA Rectum	PAP	WDA	APR	ELE	-
9.	Saraswathy	50	F	19142	CA Rectum	PAP	WDA	APR	ELE	-
10.	Sivakumar	45	M	56752	CA Rectum	ULC	WDA	APR	ELE	ADJ
11.	Bathrasammi	70	M	30099	CA Rectum	PAP	WDA	HP	EME	ADJ
12.	Kambaiyah	70	M	32917	CA Rectum	PAP	WDA	HP	EME	ADJ
13.	Kalidass	56	M	34397	CA Ceacum	ULP	WDA	RHC	ELE	ADJ
14.	Natarajan	65	M	41368	CA Rectum	INF	WDA	APR	ELE	ADJ
15.	Balagurusamy	46	M	42600	CA Hepatic Flexure	ULC	WDA	RHC	ELE	ADJ
16.	Palaniammal	57	F	45225	CA Anal Canal	ULP	PDS	-	-	CRT
17.	Karpagam	45	F	46194	CA Rectum	PAP	MDA	APR	ELE	ADJ
18.	Kalpana	23	F	40392	CA Rectum	PAP	MDA	APR	ELE	ADJ
19.	Subbathal	60	F	40464	CA Hepatic Flexure	ULC	MDA	RHC	ELE	ADJ
20.	Dhanalakshmi	38	F	56443	CA Rectum	ULC	WDA	APR	ELE	ADJ
21.	Rathinam	63	F	22802	CA Ascending Colon	ULP	WDA	RHC	ELE	ADJ
22.	Saraswathy	48	F	32763	CA Ascending Colon	TUB	MDA	RHC	ELE	ADJ
23.	Noorjahan	65	F	50971	CA Anal Canal	ULP	PDS	-	-	CRT
24.	Natchi Muthu	60	M	51477	CA Rectum	INF	WDA	APR	ELE	ADJ
25.	Iqbal	46	M	309506	CA Ceacum	ULP	WDA	RHC	ELE	ADJ
26.	Lakshmi	55	F	30672	CA Anal Canal	FUG	PDS	-	-	CRT
27.	Thangathai	65	F	481705	CA Ceacum	PRL	MDA	-	EME	ADJ

Sl.No.	Name	Age	Sex	IP. No	Diagnosis	Type of Growth	HPE	Type of Resection	Emergency / Elective	CT / RT
28.	Rathinasamy	39	M	69436	CA Rectum	PAP	WDA	APR	ELE	ADJ
29.	Marimuthu	25	M	65308	Growth Ileum	PRL	WDA	SRA	ELE	ADJ
30.	Chellaiah	62	M	63090	Splenic Flexure Growth	PRL	WDA	LHC	ELE	ADJ
31.	Rajam	46	F	60531	Splenic Flexure Growth	PRL	WDA	SRA	EME	ADJ
32.	Vellaisamy	45	M	7508	CA Rectum	PAP	WDA	APR	ELE	ADJ
33.	Angal	48	F	43159	CA Rectum	PAP	MDA	APR	ELE	ADJ
34.	Karpagam	55	F	34990	CA Rectum	PAP	MDA	APR	ELE	ADJ
35.	Saraswathy	40	F	60689	CA Descending Colon	ANN	MDA	SRA	ELE	ADJ
36.	Arumugam	60	M	51023	CA Rectum	ULC	MDA	HP	EME	ADJ
37.	Mary	57	F	440124	CA Trans. Colon	ULP	MDA	RHC	ELE	ADJ
38.	Solai	57	M	45530	CA Rectum	PAP	MDA	APR	ELE	ADJ
39.	Thangam	56	F	25877	CA Anal Canal	FUG	PDS	-	-	CRT
40.	Damayanthi	60	F	25823	CA Rectum	PAP	MDA	APR	ELE	ADJ
41.	Muthu	62	M	7488	CA Descending Colon	ANN	MDA	SRA	ELE	ADJ
42.	Pushparaj	49	M	187	CA Rectum	PAP	MDA	HP	ELE	ADJ
43.	Kondathal	55	F	23065	Growth Jejunum	PRL	MDA	SRA	EME	-
44.	Kalidas	56	M	34397	Growth Ileum	ULP	MDA	SRA	EME	-
45.	Ramasamy	60	M	20228	CA Anal Canal	ULP	PDS	-	-	CRT
46.	Patrasamy	70	M	16221	CA Anal Canal	FUG	MM	-	-	CRT
47.	Rajendran	59	M	53192	CA Anal Canal	ULP	PDS	-	-	CRT
48.	Marudhachalam	54	M	46841	CA Descending Colon	ANN	MDA	SRA	EME	-
49.	Sandammal	38	F	73263	CA Rectum	PAP	MDA	APR	ELE	ADJ
50.	Thandapani	51	M	55094	CA Anal Canal	ULP	PDS	-	-	CRT
51.	Paramasivam	60	M	59117	CA Anal Canal	ULP	PDS	-	-	CRT
52.	Karuppathal	37	F	36696	CA Rectum	PAP	MDA	APR	ELE	ADJ
53.	Parvathiammal	60	F	7626	CA Rectum	ULP	MDA	HP	ELE	ADJ
54.	Velu	61	M	41678	Growth Ileum	PAP	MDA	SRA	EME	-

Sl.No.	Name	Age	Sex	IP. No	Diagnosis	Type of Growth	HPE	Type of Resection	Emergency / Elective	CT / RT
55.	Shanmugam	59	M	1527	CA Rectum	PAP	MDA	APR	EME	-
56.	Venkatasamy	72	M	10011	CA Rectum	INF	MDA	APR	ELE	ADJ
57.	Palanisamy	55	M	16297	CA Rectum	PAP	MDA	APR	ELE	ADJ
58.	Chellappan	70	M	23100	CA Rectum	ULP	MDA	APR	ELE	ADJ
59.	Alexander	35	M	36566	CA Rectum	PAP	MDA	HP	ELE	ADJ
60.	Mohammed Sarva	58	M	40487	CA Anal Canal	ULP	PDS	-	-	CRT
61.	Velusamy	75	M	23324	CA Rectum	PAP	MDA	APR	ELE	ADJ
62.	Malliga	36	F	49997	CA Rectum	PAP	MDA	APR	ELE	ADJ
63.	Mangalam	38	F	20218	CA Rectum	ULC	MDA	APR	ELE	ADJ
64.	Manickammal	57	F	26083	CA Trans. Colon	ULP	MDA	SRA	EME	-
65.	Vellaiammal	55	F	21346	CA Descending Colon	ULP	MDA	LHC	ELE	ADJ
66.	Nagammal	60	F	21400	CA Descending Colon	ANN	MDA	LHC	ELE	ADJ
67.	Subramani	57	M	22154	CA Anal Canal	ULP	PDS	-	-	CRT
68.	Kandasamy	55	M	25413	CA Sigmoid Colon	ANN	MDA	SRA	EME	-
69.	Alagiri	72	M	60400	CA Ascending Colon	ULP	MDA	RHC	ELE	ADJ
70.	Kittammal	39	F	53010	Ca Sigmoid Colon	TUB	MDA	SRA	EME	-
71.	Chinnasamy	78	M	15397	CA Anal Canal	ULP	PDS	-	-	CRT
72.	Paramasivam	39	M	20124	CA Rectum	PRL	MDA	AR	ELE	ADJ
73.	Kanagaraj	38	M	15197	Ca Sigmoid Colon	ANN	MDA	HP	ELE	NDJ
74.	Thangadurai	40	M	60419	Ca Sigmoid Colon	ANN	MDA	SRA	ELE	-
75.	Lichmon	58	M	21606	Ca Sigmoid Colon	TUB	MDA	SRA	ELE	-
76.	Lakshmanan	40	M	26308	Ca Sigmoid Colon	ANN	MDA	AR	ELE	ADJ
77.	Manickavel	40	M	40542	CA Trans. Colon	PRL	MDA	SRA	ELE	-
78.	Palaniammal	58	F	48445	Ca Sigmoid Colon	ANN	WDA	AR	ELE	ADJ

ABBREVIATIONS :

ULC - Ulcerative; ULP - Ulcero proliferative; PRL - Proliferative; TUB - Tubular; ANN - Annular; PAP - Papliferous; INF - Infiltrative; FUG - Fungating; PDA - Poorly differentiated Adeno CA; WDA - Well differentiated Adeno CA; MDA - Moderately differentiated Adeno CA; PDS - Poorly differentiated Sq. Cell CA; MM - Malignant Melanoma; APR - Abdomino perineal resection of rectum; AR - Anterior Resection; RHC - Right Hemicolectomy; LHC - Left Hemicolectomy; SRA - Segmental Resection and Anastomosis; HP - Haatmann's Procedure; ELE - Elective; EME - Emergency; ADJ - Adjuvant; CT/RT ; NDJ - Neo Adjuvant CT ; CRT - Chemoradiation;